Modern Concepts of Cardiovascular Disease

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WHAT HAS THE ELECTROCARDIOGRAPH TO OFFER TO THE GENERAL PRACTITIONER?

It is the purpose of this article to give some idea of the sort of questions, arising in connection with the diagnosis and treatment of cardiac disease, that an electrocardiographic examination may be expected to answer or to help in answering.

In the differentiation of the various types of cardiac arhythmia the electrocardiograph is pre-eminent. Of course, after some experience has been acquired, it is not difficult to distinguish most of these disorders from each other without instrumental aid. The writer has found, however, that few master this field of diagnosis who have not either employed some graphic method of recording the heart beat themselves, or at least frequently compared the signs elicited at the bedside with graphic records. Furthermore, the recognition of auricular flutter and the differentiation of auricular fibrillation from multiple extrasystoles, of auricular from ventricular paroxysmal tachycardia, or of regularly occurring extrasystoles from partial heart-block is often difficult, if not impossible, without the aid of an electrocardiogram.

The electrocardiogram is very frequently of great value in the detection of myocardial disease. It is of most value in those cases in which the physical examination is entirely negative. One does not need an electrocardiogram to tell him that there is something wrong with the heart muscle when the heart is greatly enlarged or when the patient is confined to bed with advanced congestive heart failure. There are many instances, however, where the symptoms suggest that the heart is at fault, but where examination of the heart reveals nothing at all, or nothing more than questionable enlargement or some slight modification of the heart sounds. As a general principle, one should hesitate to ascribe such symptoms as shortness of breath, palpitation, rapid heart action, fatigue, or chest pain that is not typically anginal, to heart disease unless it can be demonstrated that the heart is abnormal. Failure to bear this in mind is likely to result in the diagnosis of cardiac disease when the real trouble is primary or secondary anemia, a pulmonary lesion, or perhaps a psychoneurosis. In such cases a distinctly abnormal electrocardiogram (the nature of the abnormality is of relatively little consequence) greatly increases the probability that no mistake will be made in attributing the symptoms to myocardial damage.

In the course of an attack of rheumatic fever or chorea, less often in the course of, or during recovery from, some other acute febrile illness such as diphtheria, the electrocardiogram may show evidence of myocardial involvement and thus lead to a guarded convalescence, when other signs that the disease has attacked the heart are lacking.

In essential hypertension changes in the heart are frequently detected by the electrocardiograph before cardiac symptoms or physical signs develop. Such changes are of value in judging whether an elevation of blood pressure has been present for some time and is likely to prove serious, or whether it is of recent origin and possibly transient.

In cases of suspected angina pectoris an abnormal electrocardiogram substantially increases the probability that the tentative diagnosis is correct, but a normal curve cannot be taken to mean that it is wrong. Sometimes changes in the form of the electrocardiographic deflections occur during an attack, furnishing clear evidence that the pain is of coronary origin.

In coronary thrombosis the electrocardiogram is very frequently characteristic, and as a rule, is far more helpful, than the physical examination of the heart which is often entirely negative, particularly when a period of ten days or more has elapsed since the onset of the attack. The electrocardiographic changes sometimes persist for years after the recurrence of myocardial infarction, and there are frequent instances in which a positive diagnosis cannot

be made without the aid of an electrocardiogram. In rare cases the electrocardiogram furnishes the only available evidence that infarction has taken place. On the other hand, a normal tracing, or one that is abnormal but not characteristic, cannot be used as a basis for excluding coronary thrombosis from consideration when the other evidence is unequivocal. Some of the most distinctive electrocardiographic changes that occur in coronary occlusion are uniformly transient and pass through a more or less definite cycle. Frequent curves are, therefore, useful in judging the progress of the condition and in deciding what liberties in the way of activity the patient should be allowed.

It is desirable, whenever possible, to control the administration of digitalis, where this drug is given rapidly or continued to the limit of tolerance, by frequent electrocardiographic examinations. Digitalis is a potent cardiac poison, and clinical symptoms and signs that one is proceeding too far are sometimes lacking or are too trivial to attract attention. Auriculoventricular dissociation with a rapid ventricular rate, or ventricular tachycardia with variation in the form of the ventricular complexes, are danger signals that may prevent fatal poisoning and may occur when there has been no vomiting or when the stage of intoxication in which it usually takes place has been passed. No patient with cardiac failure and a rapid ventricular rate should be given large doses of digitalis intravenously or intramuscularly until an electrocardiogram has been taken to make certain that the tachycardia is not of the type that would be made worse by digitalis.

The electrocardiographic control of intensive treatment of auricular flutter or fibrillation with quinidin is almost imperative.

The electrocardiogram is occasionally of value in the diagnosis of certain valve lesions, in detecting advanced mitral stenosis when the characteristic murmur is inaudible or indistinct because of cardiac failure, or in the differentiation of pulmonary stenosis from other congenital malformations and from aortic sclerosis or stenosis.

The electrocardiogram furnishes very little information regarding the functional condition of the heart, that is to say, in the diagnosis of myocardial weakness or threatened cardiac failure; nor does it offer any material aid in prognosis or in arriving at an etiological diagnosis. In young individuals with great cardiac enlargement due to congenital malformation of the heart or to rheumatic aortic insufficiency, extremely tall electrocardiograms are common and suggest that the heart muscle is in good condition. Very small electrocardiograms in patients with large hearts and cardiac failure are, as a rule, associated with a poor outlook. Intraventricular block is very much more common in arteriosclerotic and syphilitic heart disease than in rheumatic or thyrotoxic heart disease.

On the whole, therefore, electrocardiography can differentiate the arhythmias very satisfactorily, it can detect myocardial disease at times when all other methods fail, it occasionally helps in the diagnosis of valvular and congenital heart disease, but it cannot be used entirely to exclude the diagnosis of organic heart disease.

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SELECTED ABSTRACTS

Harrison, T. R., Ashman, R., and Larson, R. M.; Congestive Heart Failure; Relation Between Thickness of Cardiac Muscle Fiber and Optimum Rate of Heart. Arch. Int. Med. 49: 151: (Jan.) 1932.

In another study on congestive heart failure Harrison and others found that in their normal subjects (rat, guinea pig, rabbit, sheep, dog, man and cow) there was an inverse proportion between the thickness of the ventricular muscle fiber and the heart rate, that is, the thicker the fiber, the slower the rate. Now in patients who showed at autopsy hypertrophy of the heart muscle, the average pulse rates during life were found to be much faster, when compared with these normal subjects, than would appear to be "optimal" for a heart with such thickened fibers. The study suggests:

1) That "cardiac fatigue" in patients with large hearts may be due in part to a heart rate which is too fast. Such a rate will not allow for adequate oxygen diffusion through the thickened heart fibers during relaxation (diastole).

2) That the comparative rarity of congestive heart failure in chronic heart block may be attributed to the slow rate which approximates more nearly the optimal rate.

Steele, J. Murray; A Report of Two Cases of Localized Pleural Effusion in Heart Failure. Am. Heart Jour. 7: 212: (Dec.) 1931.

Steele reports two cases of localized pleural effusion which occurred during the course of congestive heart failure. In the first instance, the fluid which was encapsulated between the right upper and middle lobes, disappeared in eleven days coincident with improvement in the patient's condition, but subsequently reappeared with a second attack of heart failure. At autopsy there was obliteration of the right pleural cavity by adhesions, but between the upper and middle lobes there was a closed cavity containing 250 cc. of a straw-colored transudate. The second patient exhibited the same clinical features with an interval of eight years between the two attacks of failure. On both occasions the fluid disappeared as the patient improved. These cases illustrate that patients with pleural adhesions who develop cardiac failure may have encapsulated pleural effusion which will clear up without tapping as improvement takes place.

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NOTES